trointestinal infection with *M tuberculosis*. The three main proposed routes that may all play a part are direct ingestion, hematogenous spread, and lymphatic spread. Lymphatic spread would occur by transluminal transport of the tubercle bacillus to the submucosal lymph nodes with subsequent colonization and the formation of granulomas.[7,8,12,15] This theory is supported by the fact that most cases of gastrointestinal tuberculosis occur in areas rich in lymphatics, such as the ileocecal region.[7,9] Our case appears to substantiate this theory because the submucosal gastric biopsy specimen contained lymphoid follicles with germinal centers adjacent to the granulomas. It is interesting to note that often gastrointestinal tuberculosis develops in immigrants, like the patient presented here, several years after they leave their country.[12,13,16] It has been hypothesized that the infection remains dormant in the mesenteric lymph nodes with reactivation and spread at a later date.[12]

Tuberculous involvement of the stomach has been reported to occur in 0.5% to 3% of all cases of gastrointestinal tuberculosis and is often due to pulmonary infection.[9,12,13,17] Isolated gastric tuberculosis is even rarer, with to our knowledge only ten other cases having been reported in the English-language literature over the past decade.[4,5,11-13,15,17-19] As with other forms of isolated gastrointestinal tuberculosis, its incidence is probably increasing. As the case we describe here typifies, there is no pathognomonic clinical feature. Common symptoms include fatigue, weight loss, fever, and abdominal pain.[3] Laboratory findings are equally nonspecific and can show anemia and an elevated sedimentation rate.[13,14] Tuberculin skin tests are positive in about 65% of cases.[2,13] Radiographic studies may show an ulcer or a submucosal mass, most commonly in the antrum or pylorus.[3,4] Lesions may be ulcerative, hypertrophic, or a combination of the two, called ulcerohypertrophic.[3,4,7] On endoscopic or surgical biopsy, granulomas are generally present in the submucosa and subserosa, with a nonspecific inflammatory reaction in the mucosa.[7] The clinical diagnosis is often aided by the presence of caseation necrosis in the granulomas, but a definitive diagnosis usually requires the histologic or bacteriologic identification of *M tuberculosis*.[13,16] Early articles suggested that 10% of cases of gastric tuberculosis were associated with gastric carcinoma, but this correlation has not been verified in recent data.[19]

The treatment of gastric tuberculosis is similar to that of pulmonary tuberculosis.[20] Short-course treatment (six or nine months) appears to be effective, with a rapid resolution usually seen in symptoms, fevers, and the size of the gastric mass or ulcer.[19,21,22] Early suggestions that surgical extirpation of the gastric lesions is essential for cure are no longer substantiated, unless acute complications are present.[5,10,19,20] The patient in this case had a dramatic response to antituberculous treatment alone, with complete resolution of his fevers within two weeks and a rapid diminution in the size of his gastric mass.

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Mouse Joint—Another Manifestation of an Occupational Epidemic?

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IT IS AN OCCUPATIONAL epidemic that goes by many names: repetitive strain injury, overuse syndrome, or, because it usually affects the shoulder, arm, wrist, and hand, it has been called the chronic upper limb pain syndrome. Although not new, having been accurately described by Velpeau in 1840,[1(p94)] repetitive strain injuries have increasingly been recognized in the past one to two decades. There are skeptics who believe that generous workers' compensation systems and the social acceptability of repetitive strain injuries have fostered the emergence of the epidemic, both in the United States and worldwide (J. Kavanaugh, "Keyboard Cripples: The Avalanche Looms," *Business Review Week,* November 17-23, 1984, pp 37-52).[2] Indeed, studies that attempt to systematically categorize diagnoses of these injuries show that, in most instances, specific and accurate clinical diagnoses are not made.[3,4] Miller and Topliss reported that in 229 patients with repetitive trauma, 200 were given the diagnosis of chronic upper limb pain syndrome and that only 29 had specific soft tissue or rheumatologic disorders identified[5]: fibrositis (15), rotator cuff syndrome (3), mild rheumatoid arthritis (3), cervical referred pain (3), lateral epicondylitis (2), de Quervain's tenosynovitis (1), carpal tunnel syndrome

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(1), and psoriatic arthritis (1). The authors pointed out that these 29 patients should likely not have been given the diagnosis of repetitive strain injury in the first place.

Overuse syndromes and repetitive strain injuries are associated with many different kinds of activities including athletics,[5-8] sign language interpreting for the deaf,[9] the use of musical instruments[10-13] and video games,[14] and many different employment and occupational settings.[15,16] Carpal tunnel syndrome is the most commonly recognized specific repetitive strain injury. The State of Washington Department of Labor and Industries estimates that symptoms of carpal tunnel syndrome will develop in 10% of all employed adults in Washington sometime during their employment careers. The great variety of overuse syndromes has produced colorful-sounding names: Nintendo neck,[14] guitar nipple,[17] cello scrotum,[18] and potato harvester's foot drop.[19] That the names for the disorders are interesting, however, should not trivialize their importance. Many result in patient discomfort and loss of work productivity, especially when the mechanism of injury is unknown or when the offending causative agent is widespread in our society, as described in the following case.

Report of a Case

The patient, a 40-year-old left-handed man, saw his physician because of dull, persistent aching pain in the area of his second and third metacarpophalangeal joints of his right hand. The patient, an accountant, said the pain had been gradually increasing for two or three months. It was not associated with trauma, new activity, or underlying bone or joint disease. Gripping made the pain worse, and he had noticed decreased strength in the hand. He had no other painful or swollen joints or medical problems.

Examination of his hand showed tenderness and mild swelling at the base of his second and third fingers, worse on the palmar side than on the dorsum. There were no nodules, the flexor tendons were not thickened, the skin showed no erythema, and there was no joint effusion or motion limitation.

The patient was diagnosed as having flexor tendinitis and was treated with a regimen of ibuprofen, rest, and a splinting device. His symptoms resolved within four weeks. He later returned with a recurrence of his symptoms, adding that his wife had the same problem develop in the same area of her right hand. Additional history obtained at that time made the diagnosis obvious. It has remained a problem for him, though, because of his unwillingness to change his computer equipment at work.

Discussion

The patient and his wife were suffering from a heretofore-unreported condition: mouse joint. They both extensively used personal computers at work and at home that were equipped with mouse operating devices. The hand pain was likely the result of repetitive flexion activity of the index and middle fingers as required for mouse operation. "Mouse joint," it is emphasized, should not be confused with "joint mouse," the commonly used orthopedic and radiologic term that describes the small fibrous or cartilaginous loose bodies found in synovial cavities.

The mechanism of injury for mouse joint is assumed to be like that of most overuse injuries: repetitive use with resultant microtrauma and the accumulation of metabolic products causing local inflammation, an inhibition of function, and pain. There is disagreement as to the precise terms that should be used to describe such syndromes. In the United States, England, and Scandinavian countries, inflammation is the favored cause; thus, the diagnoses "tendinitis" and "tenosynovitis" are used. In Australia, the term "repetitive strain injury" is preferred,[20] proponents citing the absence of verifiable disease in most circumstances.

Regardless of the exact cause and term, mouse joint, in this patient, exhibited many of the features of other overuse injuries: its onset was gradual, it was confined to one anatomic region, it occurred in the absence of other rheumatologic conditions, it responded to conservative treatment measures, and it recurred upon return to the offending activity. Cohn and co-workers characterized repetitive strain injury in the upper extremities of interpreters for the deaf and found that pain tended to recur after two uninterrupted hours of signing and that the average number of hours worked weekly by symptomatic interpreters was 25 hours, with a range of 9 to 40 hours per week.[9] These were not new interpreters—the average number of years worked as an interpreter was 10.8.

The treatment of all overuse injuries is essentially the same, and, except for those unusual cases that require surgical intervention where improved techniques have been developed, there has been little change through the decades. Conservative measures include rest, the use of nonsteroidal anti-inflammatory drugs, splinting, steroid injections, ice massage, physical therapy, work-site alterations, relaxation training, and the evaluation and modification of biomechanical problems.

If, indeed, repetitive strain injuries are the new "occupational epidemic," it would be beneficial to recognize the injuries early and to intervene promptly, with the hope of minimizing suffering and loss of productivity. Conversely, physicians would do well not to label a simple soft tissue complaint as an ominous-sounding repetitive strain injury if there is little to support the diagnosis and if, in so doing, they unintentionally promote incapacity in patients.

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Cisplatin Therapy-Associated Recurrent Toxic Shock Syndrome

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THE IN VITRO RELATIONSHIP of magnesium to the production of toxic shock syndrome toxin-1 (TSST-1) has been previously described.[1-3] The in vivo relationship of systemic hypomagnesemia to the development of the toxic shock syndrome, however, has not been previously reported. We report a case of recurrent toxic shock syndrome following hypomagnesemia that was induced by cisplatin chemotherapy.

Report of a Case

The patient, a 46-year-old woman with non–insulin-dependent diabetes mellitus, presented with an acute abdomen. During an emergency laparotomy, an 11-cm ruptured "chocolate" cyst of the left ovary was found. Microscopic examination revealed a well-differentiated endometroid adenocarcinoma of the ovary. Primary uterine malignancy was excluded. The postoperative course was complicated by superficial wound infection, necessitating opening and draining.

A week later chemotherapy with cisplatin, 100 mg per m^2, and cyclophosphamide, 600 mg per m^2, was initiated. The patient felt well and was afebrile; serous discharge was noted in areas of wound separation. A wound specimen was obtained for culture.

The night the patient was discharged home from chemotherapy, fever accompanied by weakness, vomiting, and severe myalgia developed. In the emergency department, a diffuse erythematous rash was obvious. Her blood pressure was 84/50 mm of mercury in the supine position, with a pulse of 120 per minute and a temperature of 39.5°C. Conjunctivitis with purulent discharge and pharyngeal erythema with a strawberry tongue were noted. On abdominal examination, several areas of purulent wound discharge were found; otherwise, the findings were normal. Although lethargic, the patient was easily aroused and neurologic testing was normal.

Laboratory tests elicited the following values: glucose 14.9 mmol per liter (normal 3.85 to 6.05); sodium 131 mmol per liter (normal 135 to 148); potassium 3.5 mmol per liter (normal 3.5 to 5.3); chloride 100 mmol per liter (normal 98 to 106); bicarbonate 19 mmol per liter (normal 23 to 30); creatinine 100 μmol per liter (normal 53 to 124); urea nitrogen 3.2 mmol per liter (normal 2.2 to 8.2); leukocyte count 28×10^9 per liter (28,000 per μl) with neutrophils 0.69 (normal 0.40 to 0.74) and segmented forms 0.29 (normal 0 to 0.05); hemoglobin 109 grams per liter (normal 120 to 160); platelet count 377×10^9 per liter (normal 150 to 350); uric acid 330 μmol per liter (normal 140 to 355); lactate dehydrogenase 3.75 μkat per liter (normal 1.33 to 4.03); aspartate aminotransferase 0.58 μkat per liter (normal 0.084 to 0.67); alkaline phosphatase 1.5 μkat per liter (normal 0.64 to 1.93); bilirubin 18 μmol per liter (normal 1.7 to 24); total protein 65 grams per liter (normal 60 to 80); albumin 35 grams per liter (normal 32 to 50); calcium 2.17 mmol per liter (normal 2.13 to 2.55); and phosphorus 0.48 mmol per liter (normal 0.8 to 1.5). The prothrombin time was within normal limits; fibrin degradation products, negative; fibrinogen 3.2 grams per liter (normal 1.7 to 4.5). No infiltrate was seen on chest roentgenogram. Arterial blood gas determinations done with the patient receiving 4 liters of oxygen per minute by nasal cannula were pH 7.34 (normal 7.35 to 7.45); Po_2 11.2 kPa (normal 10.6 to 13.3); and Pco_2 3.8 kPa (normal 4.7 to 6.0). Abdominal wound culture done before admission was positive for *Staphylococcus aureus*. The serum magnesium level was not measured during this admission.

Initial therapy comprised fluids, pressor support, and broad-spectrum antibiotic coverage including antistaphylococcal therapy with vancomycin hydrochloride, later adjusted to nafcillin sodium according to culture sensitivity. Blood cultures were negative for pathogens, and eight days after admission the patient was discharged on a course of oral dicloxacillin sodium. On outpatient follow-up, desquamation of the palms and soles was noted. Assay of the *S aureus* from the wound specimen cultured during her hospital stay returned positive for the production of TSST-1.

Nine days after discharge, the patient returned for her second outpatient chemotherapy course of cisplatin and cyclophosphamide. She was afebrile, and her abdominal wounds were clean. The day after chemotherapy was given, the patient again presented to the emergency department with fever, rigors, myalgia, prostration, and vomiting. On examination she was febrile and hypotensive, with a generalized rash and purulent conjunctivitis. Her abdominal incision wounds had a purulent discharge.

The following laboratory values were obtained: glucose 11.4 mmol per liter, sodium 132 mmol per liter, potassium 2.8 mmol per liter, chloride 98 mmol per liter, bicarbonate 20 mmol per liter, calcium 2.02 mmol per liter, magnesium 0.37 mmol per liter (normal 0.7 to 1.0); and liver function studies normal; the leukocyte count was 27×10^9 per liter (27,000 per μl) with neutrophils 0.80 and segmented forms 0.17. A urinalysis showed trace proteinuria. Coagulation studies were normal. Gram-positive cocci were seen on a specimen taken from the wound.

Therapy with fluids, pressor support, and vancomycin was begun. Thrombocytopenia developed on the third day of her hospital stay, with a platelet count nadir of 26×10^9 per liter. Platelets were replenished before extensive surgical wound debridement, but no gross bleeding occurred and the thrombocytopenia resolved. Blood cultures were negative for pathogens, and antibiotic therapy was switched to a cephalosporin, to which the wound bacteria were sensitive. Desquamation of her palms and soles was seen after two weeks of hospital stay. Three weeks after admission, a third course of chemotherapy was administered with the patient continuing to receive oral cephalexin. Magnesium replacement was meticulously monitored. This course was uneventful.

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